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ORIGINAL ARTICLE

Association analysis of *MALAT1* polymorphisms and risk of psoriasis among Iranian patients

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Abstract

MALAT1 is a long non-coding transcript that affects immune reactions, thus being involved in the pathoaetiology of immune-related conditions. We investigated the associations between two genetic variants in *MALAT1* and susceptibility to psoriasis in the Iranian population. The G allele of rs619586 has been shown to be less common among cases versus controls (odds ratios (OR; 95% confidence intervals (CI)) = 0.57 (0.36–0.9)), adjusted $p = .02$). This single nucleotide polymorphism has been associated with the risk of psoriasis in a dominant model (AG + GG vs. AA: OR (95% CI) = 0.56 (0.35–0.92), adjusted $p = .04$) as well as log-additive model (OR (95% CI) = 0.59 (0.38–0.92), adjusted $p = .04$). The rs3200401 was not associated with psoriasis in any of the supposed inheritance models. This study potentiates rs619586 as a risk locus for psoriasis in the Iranian population.

KEYWORDS
lncRNA, *MALAT1*, psoriasis, rs3200401, rs619586

1 | INTRODUCTION

Psoriasis is a multifactorial condition triggered by the interplay between hereditary risk elements and environmental factors. Recent advances in genotyping methods have facilitated the identification of the genetic basis for psoriasis leading to the recognition of at least 60 susceptibility loci (Capon, 2017). Among these loci are those associated with regulation of immune responses, particularly human leucocyte antigen-C, genes regulating interleukin (IL)-23 and NF-κB signalling pathways, Tumor necrosis factor alpha (TNF-α) and genes participating in the regulation of Th2 immune response (Nair et al., 2009). Polarization of T cells towards the Th17 lineage is an important process in the pathoaetiology of psoriasis, which is particularly induced by IL-23 (Fitch et al., 2007).

A number of other biomolecules have also been reported to affect this event. For instance, *MALAT1* long non-coding RNA (lncRNA) has been shown to regulate the pattern of T-cell differentiation as its silencing has stimulated differentiation of these cells towards a Th1/Th17 and inhibited their differentiation towards a Treg phenotype (Masoumi et al., 2019). Another investigation has revealed up-regulation of *MALAT1* in serum samples of patients with psoriasis as well as their lesional and non-lesional skin samples, compared with control. Based on the observed up-regulation of *MALAT1* in psoriatic lesions, compared to non-lesional skin samples, the authors have suggested that *MALAT1* might partake in the initiation of psoriatic lesions (Elamir et al., 2021). *MALAT1* has also been shown to participate in the pathoaetiology of a number of other immune-related conditions. For instance, *MALAT1* silencing has diminished lipopolysaccharide-induced injury of primary human periodontal ligament cells by influencing the miR-769-5p/HIF3A axis (Chen et al., 2021). Moreover, the pro-inflammatory function of *MALAT1* has been proposed to partake in the hyperactive and damaging inflammatory responses during the course of Coronavirus disease 2019 (COVID-19) (Huang et al., 2021). Conversely, another study has shown that *MALAT1* silencing enhances the expression of IL-6 and Membrane cofactor protein 19 (MCP-1) inflammatory cytokines (Zhang et al., 2017).

MALAT1 has some single nucleotide polymorphisms (SNPs) with possible effects on the function or activity of this lncRNA. Among disease-associated SNPs is the rs619586, which has been found to be associated with susceptibility to multiple sclerosis (MS) in the Iranian population (Eftekharian et al., 2019). In addition, the rs3200401 of *MALAT1* has been associated with the risk of prostate adenocarcinoma in Ukrainians (Andrii et al., 2019). The T allele of rs3200401 has been associated with better clinical outcomes in patients with advanced lung cancer (J. -Z. Wang et al., 2017). Both rs619586 and rs3200401 have been associated with the risk of breast cancer in the Chinese population (Peng et al., 2018). Another study in the context of thyroid carcinoma has indicated that the G allele of rs619586 can promote cell proliferation (M. L. Wang & Liu, 2020). The G allele of rs619586 has also been shown to protect against recurrent miscarriage (Che et al., 2019). This allele has been absent among type 2 diabetes patients in an Iranian cohort (Samadi-Khouzani et al., 2021). In fact, some of the associated disorders with these polymorphisms have immunologic backgrounds implying the possible impact of these SNPs on the risk of the immune-related disorder psoriasis. Based on the association between these SNPs and mentioned disorders as well as the impact of these SNPs on cellular functions, we hypothesized that these SNPs might affect the function or activity of *MALAT1*.

The rs3200401 is located on chr11:65504361. Based on the 1000 genome project, C and T alleles of this SNP have frequencies of 85.68% and 14.32%, respectively. The rs619586 is located on chr11:65498698. The A and G alleles of this SNP have frequencies of 93.39% and 6.61%, respectively. Based on the LDpop tool (https://ldlink.nci.nih.gov/), D' statistics in different populations (based on 1000 genome project) ranged from 0.015 in Chinese Dai population to 1 in African, Europeans and many other populations.

Based on these studies that suggest the functionality of these SNPs, and the possible impact of *MALAT1* in the regulation of immune responses in psoriasis, we hypothesized that rs619586 and rs3200401 SNPs might be associated with the risk of psoriasis. So in the current investigation, we assessed the association between rs619586 and rs3200401 SNPs and the risk of psoriasis in the Iranian population.

2 | MATERIALS AND METHODS

2.1 | Enrollment of cases and controls

The current investigation was conducted on venous blood specimens gathered from 286 patients with psoriasis and 300 healthy individuals. Inclusion criteria were age ≥ 18 years and the presence of low-to-severe psoriatic plaques. The presence of other underlying diseases, pregnancy and lactation were regarded as exclusion criteria. Control persons were matched with cases in terms of sex ratio and age. They were recruited from a routine health assessment programme. They do not have any signs or symptoms of systemic or skin problems. Patients were enrolled from Shohadaye-Tajrish Hospital, Tehran, Iran, during 2016–2018. Those with systemic infection or cancer were excluded from the study. Tissue biopsies were assessed by a pathologist, and the diagnosis was made based on the presence of equal elongation of the rete ridges, dilated blood vessel, decrease in the thickness of suprapapillary plates, alternating parakeratosis, perivascular inflammatory infiltrates and epidermal infiltration of neutrophils. Individuals recruited in the control group had no history of autoimmune or systemic disorders. All cases and controls were recruited from Tehran province, and all were from a similar ethnic group (Fars). Informed consent forms were acquired from all cases and controls. The study protocol was confirmed by the ethical committee of Shahid Beheshti University of Medical Sciences (IR.SBMU.MSP.REC.1399.43).

2.2 | Identification of rs619586 and rs3200401 genotypes

Four millilitres of peripheral blood were obtained from psoriasis patients and controls. Genomic DNA was obtained from these samples using the standard salting-out procedure. The

rs619586 and rs3200401 SNPs were genotyped using the Amplification Refractory Mutation System (T-ARMS)-polymerase chain reaction (PCR) method as described formerly (Eftekharian et al., 2019). We used the Primer1 software (Collins & Ke, 2012) for designing primers. Primers for genotyping rs619586 SNP were as follow: Forward inner primer (G allele): 5′-CTTCCTTCAAAAGGTGGTAA ACTATACATG-3′, reverse inner primer (A allele): 5′-TTCTTGTGT TCTCTTGAGGGACCGT-3′, forward outer primer: 5′-CAAGAGTG GGTTTTCACGTTTCTAAGAT-3′ and reverse outer primer: 5′-TGAATGCAAACTACACATGCAGAAATAC-3′. The amplicons corresponding to G and A alleles have sizes of 213 and 279 bp, respectively. The amplicon generated by outer primers has 436 bp length.

Primers for genotyping rs3200401 SNP were as follows: forward inner primer (C allele): 5′-AGAGAATGCAGTTGTCTTGACTTCAGTTC-3′, reverse inner primer (T allele): 5′-GCATTTACTTGCCAACAGAAC AGAAAA-3′, forward outer primer: 5′-TTTAAAGAATTTTCCTTTGC AGAGGCAT-3′ and reverse outer primer: 5′-AAATTTCCTCAA CACTCAGCCTTTATCA-3′. The bands corresponding to C and T alleles have sizes of 201 and 217 bp, respectively. Outer primers generate a 422-bp band.

Reactions were arranged using Taq 2x red master mix (Ampliqon), 100 ng of DNA, 10 pmol of each inner primer and 1 pmol of each outer primer. PCR program consisted a primary denaturing phase at 95°C for 5 min; 35 cycles at 95°C for 30 s, specific annealing temperature for 35 s, 72°C for 1 min and an ultimate extension step at 72°C for 5 min. For rs619586 and rs3200401 genotyping reactions, we used annealing temperatures of 61 and 59°C, respectively. In order to appraise the set-up of the method, we have confirmed the results through sequencing of about 10% of samples.

2.3 | Statistical methods

SPSS ver. 22.0 (IBM) and SNPStats tool (Solé et al., 2006) were used for the accomplishment of statistical methods. Hardy–Weinberg equilibrium was assessed using SNPStats tool. Associations between rs619586 and rs3200401 SNPs and psoriasis were appraised in allelic, co-dominant, dominant, over-dominant, recessive and log-additive models using the chi-square test. Odds ratios (OR), 95% confidence intervals (95% CI), *p*-values and adjusted *p*-values (using Bonferroni correction method) were calculated. *p*-values less than .05 were considered as significant. Post hoc analysis was used for estimation of the required numbers of cases and controls to yield a power of 80 at $p < .05$ using the minor allele frequency of rs619586 (cases: 5.4%, controls: 9.2%). This step was accomplished using the Clincalc tool (https://clincalc.com/stats/Power.aspx).

3 | RESULTS

3.1 | General information about patients with psoriasis and healthy subjects

Table 1 shows demographic information of patients with psoriasis and controls.

TABLE 1 Demographic information of patients with psoriasis and controls

Variable	Patients	Controls
Female/male (no. (%))	124 (43.35%)/162 (56.64%)	125 (41.66%)/175 (58.33%)
Age (mean ± SD, year)	39.63 ± 17.6	38.14 ± 17.7
Age range (year)	15–85	15–84

TABLE 2 Exact test for assessment of accordance of genotype frequencies with Hardy–Weinberg equilibrium (*p*-values are shown)

	rs619586	rs3200401
Controls	.09	.84
Cases	.19	.24

3.2 | Distribution of rs619586 and rs3200401 genotypes among study subgroups

Distribution of rs619586 and rs3200401 in patients and healthy subjects was in accordance with the Hardy–Weinberg equilibrium (Table 2).

Post hoc analysis using the minor allele frequency of rs619586 (cases: 5.4%, controls: 9.2%) showed that the number of patients and controls necessary for a power of 80 at $p < .05$ is around 600 for each study group.

The G allele of rs619586 has been shown to be less common among cases, compared with healthy persons (OR (95% CI) = 0.57 (0.36–0.9)), adjusted $p = .02$). This SNP was associated with disease in the dominant model (AG + GG vs. AA: OR (95% CI) = 0.56 (0.35–0.92), adjusted $p = .04$) as well as log-additive model (OR (95% CI) = 0.59 (0.38–0.92), adjusted $p = .04$). rs3200401 was not associated with the risk of psoriasis in any of the supposed inheritance models (Table 3).

Linkage disequilibrium analyses for rs619586 and rs3200401 revealed that D' statistic = 0.2647 and $r^2 = .001056$. Then, we used LDpop tool (https://ldlink.nci.nih.gov/) to find D' statistics in different populations (based on 1000 genome project), which ranged from 0.015 in Chinese Dai population to 1 in African, Europeans and many other populations.

4 | DISCUSSION

MALAT1 has been shown to influence the pathogenesis of autoimmune disorders. Upregulation of *MALAT1* in MS patients has potentiated this lncRNA as a marker for this autoimmune condition (Shaker et al., 2019). Moreover, *MALAT1* has been reported to affect the amounts of alternatively spliced RNAs and circular RNAs in MS patients (Cardamone et al., 2019). The relevance of *MALAT1* with psoriasis is indicated by its role in the regulation of the pattern of T-cell differentiation (Masoumi et al., 2019) as well as the observed upregulation of this lncRNA in serum and skin samples of patients with psoriasis (Elamir et al., 2021).

TABLE 3 Association between rs619586 and rs3200401 polymorphisms and risk of psoriasis (chi-square test was used for statistical analyses)

Locus	Model	Genotype	Controls	Cases	Odds ratio	*p*-value	Adjusted *p*-value
rs619586	Allele	A	545 (90.8%)	541 (94.6%)	1	.01	.02
		G	55 (9.2%)	31 (5.4%)	0.57 (0.36–0.9)		
	Codominant	AA	250 (83.3%)	257 (89.9%)	1.00	.06	.12
		AG	45 (15%)	27 (9.4%)	0.58 (0.35–0.97)		
		GG	5 (1.7%)	2 (0.7%)	0.39 (0.07–2.02)		
	Dominant	AA	250 (83.3%)	257 (89.9%)	1.00	.02	.04
		AG + GG	50 (16.7%)	29 (10.1%)	0.56 (0.35–0.92)		
	Recessive	AA + AG	295 (98.3%)	284 (99.3%)	1.00	.27	.54
		GG	5 (1.7%)	2 (0.7%)	0.42 (0.08–2.16)		
	Overdominant	AA + GG	255 (85%)	259 (90.6%)	1.00	.04	.08
		AG	45 (15%)	27 (9.4%)	0.59 (0.36–0.98)		
	Log-additive				0.59 (0.38–0.92)	.02	.04
rs3200401	Allele	C	498 (83%)	487 (85.1%)	1	.32	.64
		T	102 (17%)	85 (14.9%)	0.85 (0.62–1.17)		
	Codominant	CC	207 (69%)	210 (73.4%)	1.00	.45	.9
		CT	84 (28%)	67 (23.5%)	0.79 (0.54–1.14)		
		TT	9 (3%)	9 (3.1%)	0.99 (0.38–2.53)		
	Dominant	CC	207 (69%)	210 (73.4%)	1.00	.24	.48
		CT+TT	93 (31%)	76 (26.6%)	0.81 (0.56–1.15)		
	Recessive	CC+CT	291 (97%)	277 (96.9%)	1.00	.92	1
		TT	9 (3%)	9 (3.1%)	1.05 (0.41–2.69)		
	Overdominant	CC+TT	216 (72%).	219 (76.5%)	1.00	.21	.42
		CT	84 (28%)	67 (23.5%)	0.79 (0.54–1.14)		
	Log-additive				0.86 (0.63–1.17)	.33	.66

Based on these clues, we assessed genotypes of two *MALAT1* SNPs in patients with psoriasis and healthy individuals. We reported under-representation of the G allele of rs619586 among cases, compared with controls. This SNP has been associated with the risk of psoriasis in a dominant way that AA genotype increases the risk of psoriasis, compared with AG + GG genotypes. The latter finding is in line with the reported upregulation of *MALAT1* in persons having AA genotype, compared with AG, GG and AG + GG genotypes (Peng et al., 2018). In contrast, another study has reported the role of G allele of rs619586 in triggering over-expression of *MALAT1* (Li et al., 2018). Thus, data regarding the effects of rs619586 SNP on the expression of *MALAT1* are not consistent.

Notably, the G allele of rs619586 SNP has been shown to increase the interplay between *MALAT1* and miR-214. Based on the results of the luciferase reporter assay, the transcription activity of *MALAT1* has been remarkably decreased by the G allele of this SNP following co-transfection with miR-214 mimic. Therefore, miR-214 has been shown to have the ability to attach to the G allele of this SNP (M. L. Wang & Liu, 2020). Notably, this miRNA has been reported to affect the pathogenesis of psoriasis. miR-214-3p has been among miRNAs with the highest degree in the network analyses of dysregulated miRNAs and their targets in plasma samples of patients with psoriasis (Xiao et al., 2020). Thus, rs619586 might affect the psoriasis course by changing the ability of miR-214 for binding with *MALAT1*.

The rs3200401 was not associated with the risk of psoriasis in any of the supposed inheritance models. linkage disequilibrium (LD) analysis revealed no LD between these SNPs in the assessed population in comparison with other Asian populations.

In brief, the current study potentiates rs619586 as a risk locus for psoriasis in the Iranian population. However, the underlying mechanism of its participation in this process should be assessed in future studies. We also suggest assessment of *MALAT1* genotype distributions in Cw6-positive and Cw6-negative patients.

Our study has limitations regarding sample size and study power, lack of in vitro assessment of the impact of SNPs on *MALAT1* function and lack of data regarding the severity score of patients with psoriasis.

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